

REPORTS ON
PUBLIC HEALTH AND
MEDICAL SUBJECTS

No. 81

Report on an
Outbreak of Enteric Fever

in the County Borough of Bournemouth
and in the Boroughs of Poole
and Christchurch

By

W. Vernon Shaw, O.B.E., M.D.

Crown Copyright Reserved



MINISTRY OF HEALTH

LONDON

HIS MAJESTY'S STATIONERY OFFICE

1937

Digitized by the Internet Archive
in 2019 with funding from
Wellcome Library

<https://archive.org/details/b31358354>

PREFATORY NOTE BY THE CHIEF MEDICAL OFFICER.

To the Right Hon. Sir KINGSLEY WOOD, M.P.,
Minister of Health.

SIR,

I beg to submit herewith a report by the late Dr. W. Vernon Shaw, on the outbreak of enteric fever in the County Borough of Bournemouth and in the Boroughs of Poole and Christchurch.

Information on the frequency of milkborne diseases is not easy to obtain. Individual cases and small outbreaks of enteric fever, scarlet fever, septic sore throat and diphtheria, due to infection by milk, may attract little or no attention and the source of infection may not be suspected; nevertheless a number of such limited outbreaks are reported from time to time in the Annual Reports of Medical Officers of Health. When, however, the population at risk is greatly increased by the modern trend to concentrate a large part of the retail trade in one combination, such as occurred at Bournemouth, Poole and Christchurch in 1936, and at Hove and Brighton in 1929, then a mishap at a single contributory farm may be catastrophic in its results, and the difficulty of search for the true source of infection is multiplied enormously.

That raw milk was the vehicle of infection in the present instance is certain, and Dr. Shaw has adduced strong evidence that the retailer's bulk supply was infected by a relatively small contribution, itself produced without apparent fault. How this milk became infected is not proved but there is strong support for Dr. Shaw's view that a connection exists between the "carrier" discovered and the events of May, 1934, and July, 1936.

The question of the possible dangers to the public health of the drinking by cows of sewage polluted water has been often debated and is referred to in some detail in the Report. It is eminently desirable that research into this problem should be made and, with the co-operation of veterinary pathologists, it is hoped to arrange for such an investigation. But even if the suggestion that the cow was the intermediary proves to be well founded, it does not dispose of the problem, since enteric fever would still remain a disease chiefly restricted to and spread by human beings. In milk outbreaks the infector is, in the majority of instances, an unwitting human carrier, and is the involuntary cause of much suffering. Already under the Public Health (Infectious Diseases) Regulations, 1927, there is power to deal with a human carrier when he has been detected, but it is then too late since his detection is usually consequent on an outbreak.

In the present state of our knowledge, where large milk supplies and commensurate risk are involved, the only practicable way to reduce the risk of such outbreaks to a minimum is by pasteurization.

It is fitting that I should make here a brief reference to the lamented death of Dr. Shaw. He was an epidemiologist of outstanding ability and great experience, bringing to bear on the problem under investigation a keen critical judgment and tireless persistence. Through his prompt action and advice, with the ready co-operation of the Local Authorities concerned, the epidemic which might have caused even more dire results was assuaged and checked. This report together with those he wrote on allied outbreaks at Chorley, Malton, Bolton-upon-Deane, Epping and other places will long serve as text books for the guidance of medical officers.

I have the honour to be,

Sir,

Your obedient Servant,

ARTHUR S. MACNALT. Y.

Whitehall, S.W.1.

April, 1937.

Dr. W. Vernon Shaw's Report on an Outbreak of Enteric Fever in the County Borough of Bournemouth and in the Boroughs of Poole and Christchurch.

On the 21st August, 1936, the Medical Officer of Health of Poole telegraphed that he had received a notification of a case of enteric fever and had reason to believe that many others might occur. He asked for assistance. An hour later the Deputy Medical Officer of Health of Bournemouth telegraphed that some cases of "suspected food poisoning enteric fever type" had been reported and that he too would like assistance. I was accordingly instructed to visit these boroughs, which I did on the 22nd August and on many subsequent occasions. Sir Arthur MacNalty, the Chief Medical Officer of the Ministry, also visited the district on 31st August and 1st September and conferred with representatives of the local authorities concerned.

At a conference at Poole on 22nd August with the medical officers of health and a number of medical men practising in Bournemouth and Poole, I learned that within the previous 24 hours some 30 cases had been notified, and that many others were under observation and were probably of a like nature. The patients were scattered throughout Bournemouth, Poole and Christchurch and it was plain that a widely distributed article of food or drink was concerned in the spread of the infection. Water suggested itself, but inasmuch as three different supplies would require to have been simultaneously involved, this explanation was regarded as improbable. Moreover, further inquiry quickly made it apparent that the only factor common to all the patients was the consumption of raw milk retailed by one distributor. According to the 1931 census the resident population at risk in the three towns was 195,367. Of this number the dealer referred to supplied approximately 10,000, apart from those who bought milk over the counter at his 11 branch depots (six in Bournemouth, four in Poole and one in Christchurch).

The fact that the total incidence of enteric fever was concentrated on this relatively small group of the population satisfied me that the milk was at fault, and measures were at once taken to render it safe. The dealer had a central depot in Poole; he employed 12 roundsmen who were responsible for the distribution of all the milk except a small quantity allowed to the staff. They delivered bottles of raw mixed milk, or of raw Jersey and Guernsey milk, to private houses and to branch depots—where it was sold over the counter—throughout the three towns; they also delivered raw mixed milk in bulk to certain hotels. I ascertained that not only was every man's round implicated but that all the milk, whether bottled or not, was involved. Provisionally, therefore, I concluded that

the roundsmen could be eliminated as the source of infection but that it was not possible to exonerate any of the milk distributed from the central depot. The business was established to supply raw milk as fresh as it could be obtained, but when the dealer was informed of the tentative conclusions I had arrived at, he fell in with the suggestion that he should distribute no more raw milk but should pasteurize* the whole supply. All the milk distributed after the morning round on 22nd August was pasteurized in the manner set out in Appendix I to this report. The total amount of milk received daily at the central depot was approximately 1,600 gallons, and it is computed that the number of regular customers (householders) was not less than 10,000, exclusive of the unknown number who bought milk at the branch depots. With pasteurization in force it was confidently anticipated that no infection would take place after 22nd August, and that therefore no notification of a *primary* case of enteric fever would be received after the expiration of the incubation period calculated from that date *plus* a week or ten days which might be expected to elapse before a doctor was called in. The charts appended show that this is, in effect, what took place. The supply having been rendered safe, an attempt was then made to discover how the milk had become infected.

PREVIOUS RECENT INCIDENCE OF ENTERIC FEVER (INCLUDING PARATYPHOID) IN THE THREE TOWNS AND IN THE REST OF COUNTY DORSET.

In the last three calendar years there were notified in Bournemouth 7 cases, 3 in 1933 and 4 in 1935; in Poole there were 10, 1 in 1933, 3 in 1934 and 6 in 1935; in Christchurch there was none.

In the County of Dorset (exclusive of Poole) there were 23, 8 in 1933 (1 in the Borough of Weymouth, 4 in the Rural District of Dorchester and 1 each in the Rural Districts of Sherborne, Wareham and Wimborne), 6 in 1934 (4 in the Rural District of Dorchester and 2 in the Rural District of Wimborne) and 9 in 1935 (2 in the Urban District of Swanage, 2 in the Rural District of Blandford and 5 in the Rural District of Dorchester).

In 1936 prior to August there were four cases in Bournemouth, two in the week ending 1st February and two in the week ending 11th July; in Poole there was one, in the week ending 30th May; in the Borough of Blandford Forum there were two, one in the week ending 7th March and the other in the week ending

* Throughout this report I have for convenience used the terms "pasteurize" and "pasteurization" in their wide popular sense. The process used, though no doubt adequate in an emergency, was not pasteurization as defined in the Milk (Special Designations) Order, 1936, and milk so treated could not lawfully be sold as "Pasteurized".

16th May; in the Rural District of Dorchester there were two, one in the week ending 4th April and the other in the week ending 9th May; in the Rural District of Blandford there was one, in the week ending 16th May. From the information obtained by the medical officers of health I concluded that the earliest patients in the outbreak under investigation were infected on or about the 20th July, and that there was no evidence to suggest that they were causally associated with any of the previously notified cases. This limited the investigation to what I call for convenience "the critical period", 20th July to 22nd August, inclusive.

PERSONNEL AND PROCEDURE AT THE DEALER'S DEPOT DURING THE CRITICAL PERIOD.

There were employed at the central depot in addition to the office staff, a foreman, a yardman, a horseman, a storekeeper, a man to wash the cooler and churns, three young women who washed bottles, and 12 roundsmen. All the milk was purchased and brought in in motor vehicles by three men drivers who twice daily collected it in churns directly or indirectly from 37 producers scattered throughout a large part of County Dorset. The individual producers' daily contributions varied from 5 to 300 gallons. When the milk arrived at the depot the contents of the churns were poured into a tank where they were strained and pumped up into another tank on the first floor. From this the milk ran over a cooler either into a bottle-filling machine, or into sterilized churns for bulk delivery. In the event of infected milk being passed through the plant, the whole of the milk subsequently passed through at the same operation was likely to become infected. Delivery was entirely by the 12 roundsmen; no milk was sent out a second time; if any was brought back undelivered, it was either scalded for clotted cream, put into a separator for creaming, or used for cheese making. All the churns were sterilized by steam before being returned to the producers and the dealer's plant was similarly treated twice a day, i.e. after use in the morning and again in the evening. The water supply was obtained from the town mains and from an artesian well, and was proved to be above suspicion.

I was unable to obtain any history of suspicious illness, either recent or remote, of any of the persons employed at the central depot. Most of them were old employees, but one was a newcomer in the place of a bottle washer whom I also saw. Furthermore, specimens of blood were taken on the 24th August from all the employees and were examined by the Widal test and found negative. I concluded therefore that the source of infection was probably to be found elsewhere and the investigation accordingly moved to the producers, but before giving the results of my later inquiries I shall describe briefly the further progress of the epidemic.

COURSE OF THE EPIDEMIC.

While the investigations at the producers' farms were in progress, enteric fever cases continued to be notified in Bournemouth, Poole and Christchurch, and at the same time many intimations were also received from medical officers of health in various parts of the country, of cases among visitors who had developed the disease after their return home. Enquiries into these further cases served only to confirm my tentative deduction of 22nd August. Raw milk from one supplier was the factor common to all the primary cases. The total number of known cases finally amounted to 718, of whom approximately 518 were residents and 200 visitors. So far as is known only four secondary cases occurred. The deaths among residents numbered 51.

I append below four charts, three of which (I., II., III.) set out for Bournemouth, Poole and Christchurch, respectively, the numbers of primary cases among residents and visitors separately, together with the approximate dates of the onset of illness in each case. Examination of the charts indicates that there is a striking similarity between the three towns, and if Christchurch with its comparatively small standing population of 13,000 and therefore low incidence (c.f. Bournemouth 125,000, Poole 67,000) be excluded, the course of events in the two larger towns is, from the epidemiological point of view, so similar in all respects as itself to be reasonably conclusive evidence that the same causal factor was at work in both. The charts relating to the visitors are instructive, as, although they exhibit a correspondence with those of the residents, the outbreak began definitely later where Poole is concerned. The influence on holiday-makers during the August Bank holiday week-end is apparent. Chart IV is a sum of the other three in as far as residents are concerned.

Most authorities agree that the incubation period of enteric fever is about 12 to 14 days. It probably varies with the dose of the infecting organisms, but the limits rarely lie outside 7 to 17 days.

The onset of the disease is slow and insidious and it is common to find that a patient has been ill for a week before medical attention is sought; frequently, therefore, three weeks or more elapse between the dates of infection and of notification. For the purposes of my inquiry an incubation period of 14 days was assumed. On this basis, which received striking confirmation from the history of a number of patients who had paid only fleeting visits to the towns, Chart V has been prepared. The earliest date on which the milk was infective was 20th July. From this date the infection, as indicated by the rapid increase of notifications, became more intense until it reached a maximum on the 6th August, followed on the 7th and 8th by a sudden drop of approximately 30 per cent. Thereafter for two days there was a rise but from the 10th August there was a decline, at first

rapid and then almost uninterrupted, over a space of a little more than three weeks when the infection finally disappeared. In interpreting this chart it should be borne in mind that the amount of milk consumed, and therefore the population at risk, increased at week-ends. It might appear that some patients were infected *after* pasteurization commenced, but I am satisfied that this is not so. Any patients in whom the arbitrary 14 days incubation period was exceeded would necessarily begin to be ill at a correspondingly later date. Moreover, some of the alleged late cases were, in fact, relapses in patients who had failed to call in medical attention in the initial illness. Although there had been no delay in pasteurization, it will be observed that when this was first done, the milk had already largely lost its infective property. From the whole evidence it was concluded that the milk was infective for a period of 31 days or thereabouts before pasteurization was adopted.

BOURNEMOUTH.

Residents.

Visitors.

41218

POOLE.

Residents.

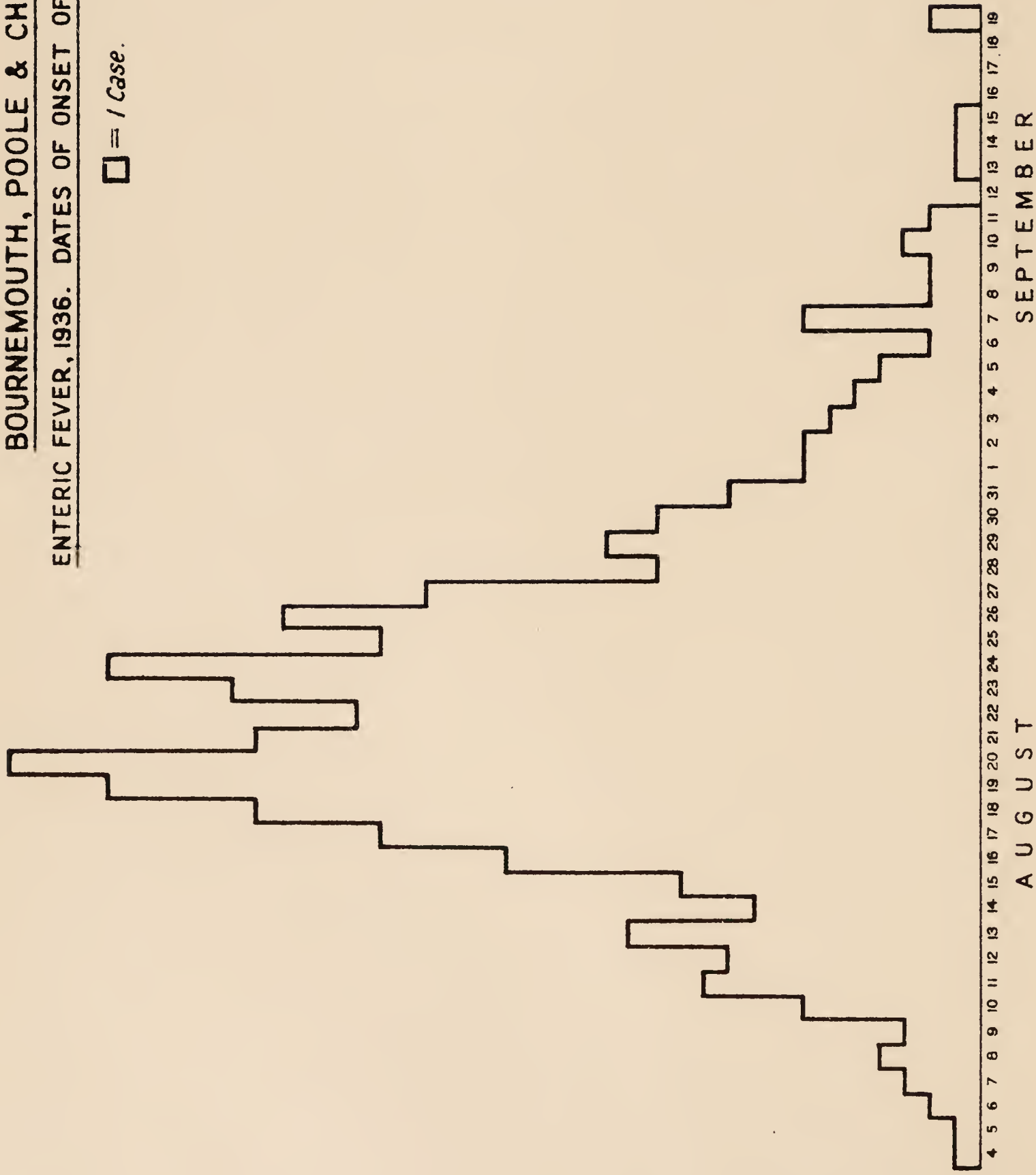
[illegible]

BOURNEMOUTH, POOLE & CHRISTCHURCH.

CHART IV.

ENTERIC FEVER, 1936. DATES OF ONSET OF ILLNESS OF RESIDENTS.

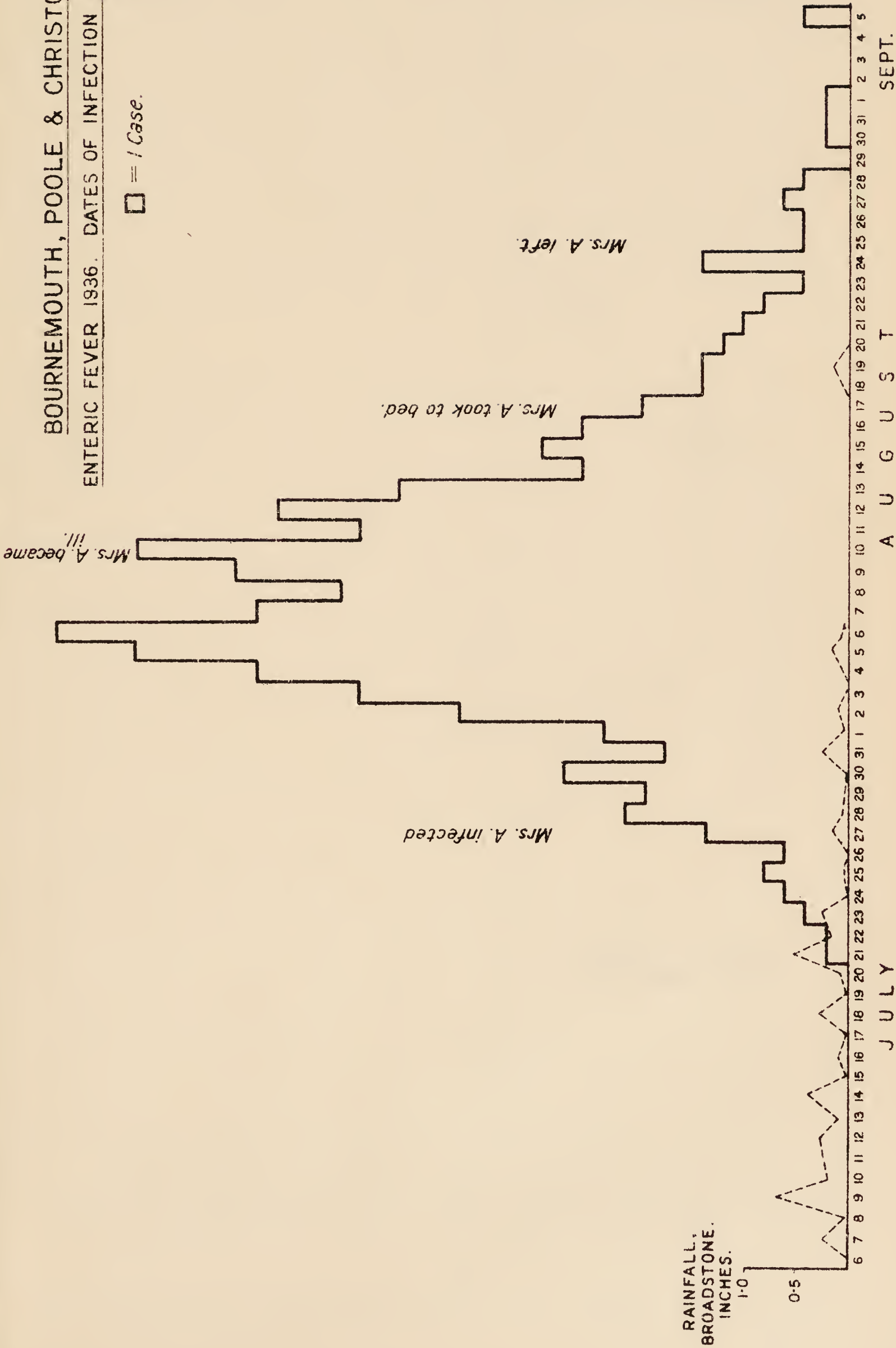
□ = / Case.

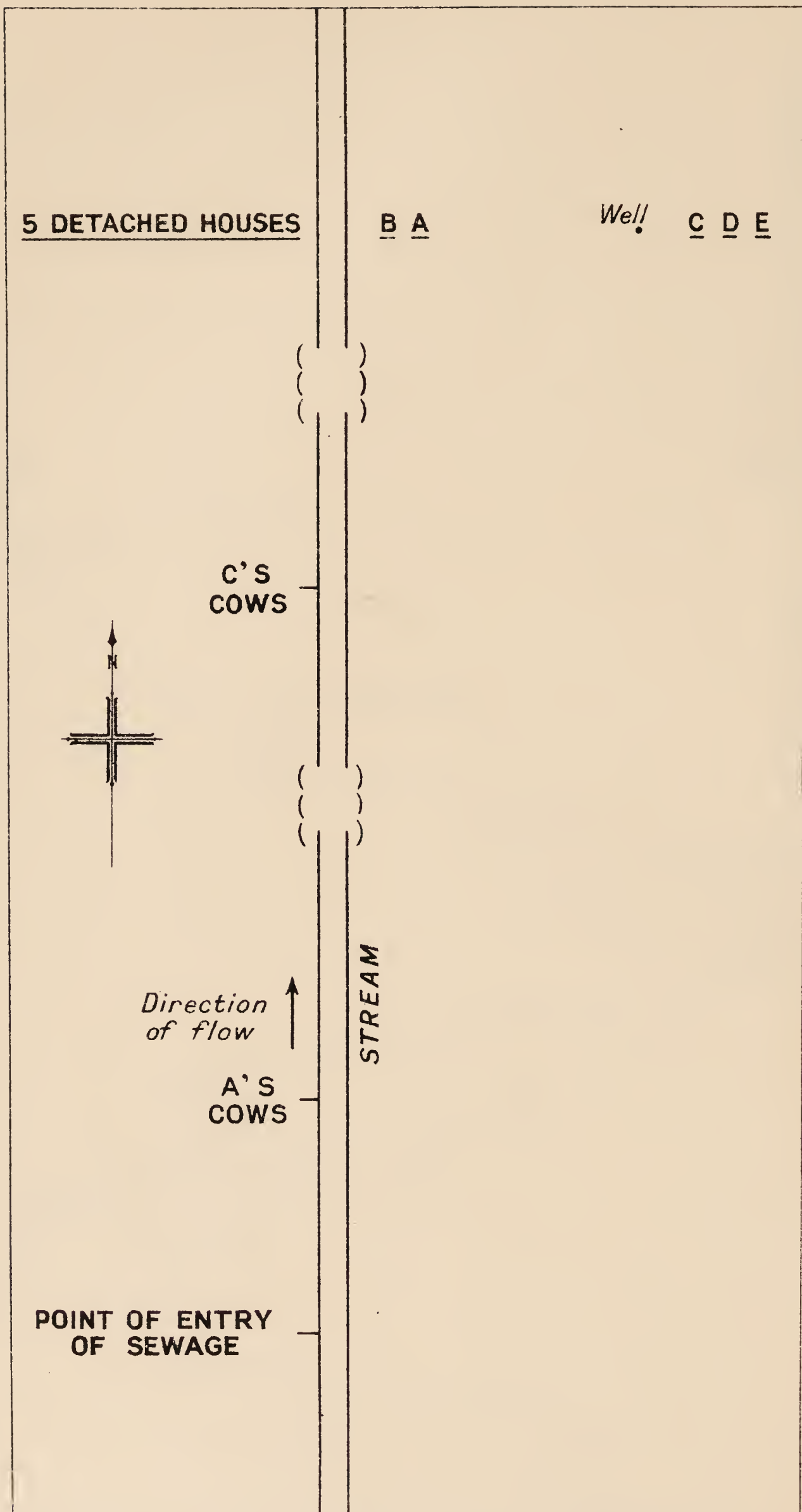


BOURNEMOUTH, POOLE & CHRISTCHURCH.

ENTERIC FEVER 1936. DATES OF INFECTION OF RESIDENTS.

□ = 1 Case.





B and A shew the relative position of the two cottages of the tenants designated by those letters. Similarly C, D & E represent the subdivision of the original farm house now occupied by the families C, D & E respectively.

B, A, C, D, E together with the cowsheds, etc., in their immediate vicinity constitute X.

INQUIRIES AT MILK PRODUCERS' PREMISES.

All the 37 producers' premises were visited and enquiries made there by my colleague Dr. Conybeare, who by this time had joined me, or by myself, into the health of such of the personnel as had had any part in the production of milk during the critical period. In all 192 persons were examined, and where necessary samples of blood were taken and examined either at the Ministry's or at one of the local public health laboratories. Only two persons gave evidence of enteric fever infection and these were both patients, Mrs. A., the wife of a milk producer at X, who supplied 20 gallons of milk a day to the dealer, and her son, a boy aged 12 years. Mrs. A. was found at Y, having been removed from X on 25th August when she was regarded as being sufficiently recovered to travel. She was subsequently removed at my suggestion to hospital where she died from enteric fever on 7th September. The position at X is indicated in the diagram opposite.

X was formerly a farm with two labourers' semi-detached cottages. Some years ago it was split up into five holdings and now, as indicated in the diagram, three separate families C., D. and E. occupy the original farmhouse and two others, B. and A., the semi-detached cottages. All the tenants were milk producers and four of them (A., B., C. and D.) supplied in the aggregate 65 gallons of milk a day, in quantities varying from 8 to 20 gallons, to the dealer concerned. E., the fifth tenant, supplied another dealer who pasteurized his milk. Since October, 1935, A. has lived in one of the two cottages with his wife and son aged 12 years. His holding consists of 26 acres and he had 11 cows in milk at the time of my visit; he did all his own farm work. His cottage is of two storeys; on the ground floor are a parlour, a kitchen, and off the kitchen a small scullery in which cold water is laid on; on the first floor are three bedrooms. Five or six yards to the north of the cottage is a one-roomed dairy (10 ft. by 9 ft.) with a concrete floor and bench; it contains no means of heating and cold water only is laid on. About the same distance north of the dairy is a small washhouse (9 ft. by 6 ft.) in which is a coal-heated washing boiler but no water. Adjoining the washhouse is a pail closet. In the adjoining premises which are similar, Mr. and Mrs. B. have lived for 15 years; their only child (I.B.), a girl of 19, was admitted from here to the Poole Isolation Hospital on 16th May, 1934, suffering from enteric fever, from which she died. East of the cottages occupied by A. and B. is a large cowshed divided up into five sections each housing the cattle of one of the tenants. East of this again is the farmhouse already alluded to and the families occupying it are C. (consisting of a man, his wife and grown-up daughter who came here in October, 1935), D. (a man and wife who

came in March, 1927), and E. (man, wife and two children who came in March, 1936). About 40 feet west of the cottages occupied by A. and B. and running in a south to north direction is a stream, perhaps 6 ft. to 9 ft. wide, which forms the garden boundary of B.'s cottage. The depth of the stream is such that in dry weather it is easily forded, but it is plain from the appearance of the banks that on occasions the depth in some places might be as much as 3 ft. On either side of the stream and to the south of the cottage lies the bulk of the land farmed by the five tenants. Into the stream, at a point half a mile south of the cottages, i.e. upstream, an opaque fluid having the appearance of sewage was found to be discharging from a 4 in. pipe which emerged from the right bank about 18 in. above the bed of the stream. The water supply for the cottages is derived from a covered well 162 ft. deep sunk in 1916, and situated 100 yards to the east of the stream at a point to the west of the original farmhouse between it and the cowsheds. The water of the well is raised by motor power to a large tank from which it gravitates to the houses and dairies of the tenants, and also to five detached houses occupied by 18 persons situated to the west of the stream and within 250 yards of the well. With the exception of Mrs. A., I was unable to learn of any recent illness at the producers' farms.

Mrs. A. began to be ill on the 10th August and it is probable that she was infected on or about the 27th July. She continued unaided to do her housework until the 17th August, on which day her father came over from Y. to see her. On the 18th August she was seen by a medical man for the first time and from this date until the 25th August, when she was removed to her father's house at Y., Mrs. E. one of her neighbours, helped to look after her during the daytime. Mrs. E. stated that Mrs. A. suffered from diarrhoea and that she passed blood by the bowel, and this statement is supported by a Mrs. K. who came to see her on the 20th August and who washed some of her soiled body-linen in a galvanized zinc receptacle which was customarily used by Mr. A. for the washing of his milking utensils. On the 24th August Mrs. A.'s medical attendant consented to her removal to her father's house. At the same time he wrote to the medical practitioner into whose care she was going suggesting that she had had influenza, but that there was an element of doubt about the diagnosis. The second practitioner saw Mrs. A. on 27th and 29th August, when her condition was not inconsistent with the diagnosis of his colleague. Mrs. A. was found at her father's house by Dr. Conybeare on the 31st August, on which day I also saw her. We agreed that she had enteric fever and after communication with the medical practitioner in attendance she was removed to hospital where she died on the 7th September. In

the meantime, Mrs. A.'s only child, a boy of 12 who had remained at X with his father, developed the disease. He was infected on or about 20th August at a time when his mother was ill in bed; he was first seen by a medical man on 3rd September and was at once removed to hospital. From the 27th July—the probable date of infection of Mrs. A.—until the 18th August, Mr. A., her husband, was constantly in contact with her by day and night and there was no one else, except her 12 year old boy, to do anything for her. Moreover, the assistance he received from the 18th to the 25th August was occasional and was limited to the day time. During this period therefore Mr. A. was called upon to do a certain amount of housework and nursing including the preparation of food, the washing up of eating and drinking utensils, and the emptying of bedroom slops, in addition to which he continued unaided to do all the work incidental to the production from his own cows of his daily quota of approximately 20 gallons of milk. His milking utensils are said to have been washed in water heated on the kitchen fire and carried to the dairy for the purpose. This may be so, but, inasmuch as it would have been easier to take these vessels into his house and wash them there, this possibility cannot be excluded.

The significance of Mrs. A.'s illness lay in the fact that although from the point of view of time and place she was but one case out of many, she was the only primary case who had not drunk the raw mixed milk under suspicion in Bournemouth and Poole. She however had access to and probably drank the raw milk produced by Mr. A., who was one of the 37 producers under investigation. My colleague, Dr. Scott, later demonstrated that the strain of *B. typhosus* with which Mrs. A.'s son was infected was identical culturally with that recovered from the Bournemouth patients; and inasmuch as he was almost certainly infected by his mother, it follows that she had the same strain.

That there is some connection between the infection of Mrs. A. and that of the Bournemouth, Poole and Christchurch patients is very probable, but the dealer's supply was first infective on or about 20th July, whereas Mrs. A. was not infected until about a week later. It appeared at first very improbable that this patient should be the sole exception, and that she alone of several hundred primary cases should not have drunk the milk distributed by the dealer. The evidence, too, that she was not causally connected, as she could not have been if the history is correct, save in most exceptional circumstances, was received with the greatest reserve, particularly because the conditions under which her husband's milk was produced were calculated to lead to its contamination once the specific infection was introduced into his household. But here again the evidence is

clear; Mrs. A.'s onset of illness was not before the 10th August by which time twenty-nine other patients had fallen ill, seven Bournemouth residents, eight visitors and fourteen Poole residents, and in this respect she belongs to the Bournemouth, Poole, Christchurch series. Certain assumptions would be necessary to justify the conclusion that Mrs. A. was the primary source of infection. These are that she was infective from the very beginning of her incubation period, that she infected the milk practically at once and consistently for several days immediately thereafter and that the incubation period of the early Bournemouth and Poole cases was not more than seven days. Such a combination of circumstances is very unlikely, although it is admitted that infectivity during the incubation period is possible. It is not rare to find the specific organism in the faeces in this stage, and indeed Conradi considered infection by patients in the incubation period to be an important factor in the spread of enteric fever.* It is said that Mrs. A. took no part in milk production during the last week in July, but this statement is to be accepted with some reserve; she occasionally assisted in carrying milk from the cowshed to the dairy, and, inasmuch as her husband was dependent on water heated on the kitchen range for the cleaning of his milk utensils, it is doubtful if Mrs. A.'s participation in production—including the washing of milk utensils—was as limited as it is stated to have been. But that Mrs. A. was primarily responsible for the outbreak is very improbable and this led to further investigations.

It is recorded above that I.B., the daughter of the A.'s next-door neighbour, died from enteric fever, probably contracted at home, in May, 1934. She had been in the habit of eating watercress gathered from the stream to which reference has been made, near her parents' house. At that time a bacteriological examination of the water supply from the well already referred to was made, and organisms of intestinal origin found present in 25 *ml.* Although I had satisfied myself that, so far as the present tenants of the five cottages and the five detached houses on the same water supply were concerned, no illness of any consequence other than those of Mrs. A. and I.B. had occurred, the fact that there had been two, apparently sporadic, cases of enteric fever in adjoining houses at an interval of two years suggested inquiry into the medical history of the previous occupants of the cottages. The information may be summarized thus:—

* Conradi, Deut. med. Wchr. 1907, XXXIII, 1684.

A's Cottage.	B's Cottage.	C's Cottage.	D's Cottage.	E's Cottage.
<p>1922—Oct. 1935. Mr. F.—Occasional diarrhoeal attacks.</p> <p>Mrs. F.—Nil. Daughter—Nil.</p> <p>Mother-in-law died aet. 85. A.—from Oct., 1935. Mr. A.—Nil. Mrs. A.—Enteric fever fatal (July, 1936). Son—Enteric fever (secondary, August, 1936).</p>	<p>1922—1935. Mr. B.—Nil. Mrs. B.—Nil. Daughter—Enteric fever, fatal, May, 1934.</p>	<p>Before 1932. The L. Family. No information.</p> <p>1932—Oct., 1935. Mr. G.—Nil. Mrs. G.—died Pul. Tub. (1935). C. from Oct., 1935. Mr. C.—Nil. Mrs. C.—Nil. Daughter—Nil.</p>	<p>1922—1927. Mr. H. died—Carcinoma (1923). Mrs. H.—Nil. Son—Nil.</p> <p>1927—36. Mr. D.—Nil. Mrs. D.—Nil.</p>	<p>1922—1935. Mr. M.—Nil.</p> <p>From March, 1936. Mr. E.—Nil. Mrs. E.—Nil.</p> <p>2 children—Nil.</p>

Samples of blood from all the adult occupants of the cottages and from Mrs. A.'s son were examined, and with the exception of those of Mrs. A. and her son the results were negative; the clinical history is consistent with these results. No history of enteric fever before May, 1934, was forthcoming, and there is no direct connection between the B. case of 1934 and the A. case of 1936. What the cause of the occasional diarrhoeal attacks were from which Mr. F. is alleged to have suffered from time to time between 1922 and 1935 can only be conjectured.

Chart V shows graphically the dates of infection of all the primary cases and on it are noted also the dates on which Mrs. A. was infected, on which she began to be ill, when she took to bed, and when she was removed. The infections were most numerous four days before she began to be ill, and 11 days before she took to bed. Unless all the susceptible milk drinkers had already been infected, which seems improbable, one would have expected, had she been even partly responsible, a further rise in the number of infections after she became ill.

It is necessary to explain the independent infection of Mrs. A. and if in so doing a plausible or even possible explanation of the illness of I.B. in 1934 is forthcoming, the evidence is thereby strengthened. Now Mrs. A. had access to her husband's milk and to no other, and it has been shown that if one portion of the dealer's milk became infected it was almost certain that the whole of his supply would be involved. In the circumstances it is reasonable to conclude that Mrs. A. was infected by her husband's milk, and there is strong presumptive evidence that this milk was also responsible for the outbreak. The only individual handling the milk was A.; he may have had some assistance from Mrs. A. before the 17th August but he had none after. So far as it is possible in the present state of our knowledge to exculpate A., this has been done, and if direct personal infection is excluded, another possible source has to be sought in an infected vehicle. Water appears to be the only likely agent, but it is necessary to show that it was specifically infected at a certain time and that it was either drunk or used in such a way as to contaminate food or milk. We know from the examination of the well water in 1934 that it then contained organisms of intestinal origin, as it also did in September and early November, 1936. The structure and topography of the well are not such as lend themselves to the contamination of the contents. Nevertheless, the presence from time to time of these organisms is suspicious, and although later examinations of the water showed that at the time the particular samples were taken it was of good potable quality, it cannot be said that it was always above suspicion. The matter will be referred to again. Moreover, it is to be remembered that this was the only water available for all purposes (including the washing of the milk utensils of four other producers besides A.) for 32 persons in ten dwellings,

whereas only one case of enteric fever occurred among them. This argument is not conclusive, yet, if the extreme infective power of the dealer's mixed milk was due to the infection of A.'s contribution and this in turn was due to specific pollution of the common well, then more cases amongst the 32 potential local water consumers would have been expected. In the circumstances I do not think that the well water was at fault.

Reference has been made to a stream which on its way to the River Stour passes through the tenants' land and skirts the garden of B.'s cottage, and to a possible source of its pollution by an effluent from a 4-in. pipe. The bed of the stream is flat and the water shallow except after rain, when the level is apt to rise considerably. Bacteriological examinations of the effluent for the presence of the *B. typhosus* on four occasions in September and early October proved negative, but on the 16th and 24th of October this organism was present in large numbers, whereas it was not to be found in the water of the stream above the point of entry of the effluent. Below that point the water contained large numbers of organisms of intestinal origin which it was of no value to differentiate.

The effluent came from a small impervious tank of 446 gallons' capacity built many years ago to take the sewage and storm water of a large house. The tank is situated about 100 yds. east of, and 12 yds. above, the stream level. A little above midway at one side of the tank, and a little below it at the opposite side, are the inlet and outlet respectively, and the position is such that once the tank has become filled to the level of the outlet any addition to its contents promptly flows through the outlet and, unless there is any obstruction, finds its way quickly to the stream.

At the time of my visit the total number of occupants of the house was 16, but from time to time the number fluctuates. The water supply to the house is from the mains of the Bournemouth Gas and Water Company; the sewage is removed by water carriage, but as the capacity of the tank is less than the average daily amount of sewage produced when the house is fully occupied there is usually no delay in its passage to the stream. Of the 16 occupants, ten had been given anti-typhoid vaccine a few weeks earlier. Nothing was to be gained therefore by blood examinations except in the case of the remaining six residents, specimens of whose blood failed to give the enteric fever reaction. Two of the occupants, whose excreta were unobtainable, gave a clean history and there is no reason to suppose that either was a carrier. Faeces and urine were obtained from the other 14 and were examined with entirely negative results, except in the case of one of them whose faeces on two occasions at five days' interval contained the *B. typhosus*. Two examinations only were made.

This individual, who was unaware that he was a carrier and was most distressed to learn that he could even remotely be associated with the outbreak, did all he possibly could to facilitate our investigation. He gave no definite history of having had enteric fever; the possibility of his having been infected during the outbreak was considered and, it is thought, can be rejected not only on clinical grounds but also from a survey of his movements at the time. But many years ago he had an acute febrile illness which nearly proved fatal and which, in view of the present bacteriological findings, was probably enteric fever. It is also suggestive that during the last three years he has been subject to periodical attacks of abdominal discomfort which are characteristic of the carrier condition. He was staying here in April, 1934, when I.B. was infected, and was here also throughout the summer of 1936 until the 10th August, when he went away and did not return until the 28th September.

Owing to a reduction in the number of occupants of this house after 10th August, only a small amount of excreta was discharged into the tank from then until late September, and it is significant that in the effluent on the 16th, 18th, 21st and 24th September and 2nd October the *B. typhosus* was not found, but it was present in large numbers on the 16th and 24th October, 18 and 26 days respectively after the suspected carrier had returned. The organism proved to be of the same cultural type as that recovered from such Bournemouth patients as were examined and also from Mrs. A.'s son. The evidence therefore shows that this person is a typhoid carrier and, as is usual in such carriers, has been excreting the *B. typhosus* intermittently over a considerable period.

We have then the position that there was, probably in April, 1934, and almost certainly in June and July, 1936, a potential source of infection at this house and that the stream into which the sewage from it flowed was little more than an open sewer liable at any time when the particular individual was staying there to contain the *B. typhosus*.

The stream rises at about O.D. + 240, in marshy ground some two miles south of the cottages. Its level at the cottages is approximately O.D. + 100, or thereabouts, and, as I have pointed out, in some places it may on occasion be 3 ft. deep. It flows through coppices and pasture land which it drains, and no other obvious source of gross human pollution was found.

The possibility of infection of A.'s milk through the agency of the water of the stream must now be considered. There are three ways in which this may have occurred—

- (1) That water direct from the stream was used for dairy purposes.
- (2) That it found its way into the common well.

(3) That A.'s cows (which were in the habit of drinking at the stream) conveyed the infection.

(1) There is no evidence of this. With regard to (2) two experiments were made. A quantity of a harmless dye (fluorescin) was put into the stream, and thereafter samples were taken from the well for a number of days without the dye being found in them. It is to be borne in mind that this test is dependent on naked eye appearances and is of value mainly where subterranean water courses are present or are suspected. The second test was more delicate. After having ascertained that there was no lithium in the well water, a quantity of lithium citrate was put into the stream and lithium was subsequently detected in the well water by means of the spectroscopic test. This is regarded as an exacting test and it does not follow that micro-organisms would behave in the same way. Nevertheless the result indicates that there is a communication from stream to well though possibly an intermittent one; but from the bacteriological evidence it would appear that the route may be impassable to pathogenic organisms or only intermittently practicable.

As to (3), Dr. Scott points out that the transmission of the infection by cows in contact with human sewage is theoretically possible in three different ways.

(a) By fouling of the udder and teats, followed by direct mechanical transfer of specifically infected material to the milk.

Reference to the diagram accompanying this report shows that both A.'s and C.'s cows had access to the stream (at places 86 yds., and a quarter of a mile, respectively), below the point of discharge of the sewage. They therefore had ample opportunity on occasions of drinking specifically infected water and of fouling their feet and legs. I have already referred to the marked fluctuations in the level of the stream, and there is no doubt that on many occasions the cows' teats and udders were liable to become wetted with infected water. This may have happened in July.

If the conclusion set out above with regard to the carrier is correct, then whenever the effluent became specifically polluted, the cows were standing in and were drinking typhoid-contaminated water. C.'s cows were exposed to a similar risk, and if A.'s cows carried the contamination there is no reason why C.'s should not. It is true that no member of C.'s family (three persons) contracted enteric fever, but they all denied drinking raw milk at any time. When A. drove in his cows to be milked, he is said to have washed their udders with cold water and a cloth, but the same water sufficed for more than one cow and the same

cloth for all of them. Of the value of perfunctory washing in such circumstances I am doubtful and if, as was possible in certain circumstances, the udder and teats did become contaminated, infection of the milk would almost certainly follow. But it is evident that the dealer's milk was infective continuously for three to four weeks, and if it was infected in this way, then it is necessary to postulate repeated surface contamination of the udder or teats day by day. That this may take place in the manner indicated will be generally admitted, but not as a regular daily phenomenon or as anything but an exceptional occurrence.

(b) By the ingestion of specifically infected material in the water, followed by excretion in the dung or urine of viable typhoid organisms and contamination of the milk during milking.

We have been unable to find any information on this head and the finding of typhoid organisms in the dung of cows has never been reported.

(c) By the systemic infection of the animal and the actual excretion of the organisms in the milk.

This has always been regarded as an impossibility, but my attention has been called to one reasonably authenticated report* of the isolation of the typhoid organism from the tissues of a cow (splenic abscess) although no information is given as to the possible sources of infection of the animal. A cow in the condition described in the report alluded to might well have excreted typhoid organisms in her milk; the excretion in milk of the biologically similar Gaertner's bacillus has been reported several times.

Either of the methods (b) or (c)—but especially (c)—would permit of the daily abundant infection of milk over a period of days or weeks, thereby differing from method (a). We are not in a position to say that infection by method (b) or (c) does occur, but on the other hand we cannot assert that it does not. It is possible that during the period of its greatest infectivity, the first two weeks in August, the milk was being specifically infected not only in the manner just suggested but also by the various imaginable contacts it had with Mrs. A. during the incubation period and early stages of her illness.

CONCLUSIONS.

1. That the outbreak was due to the consumption of raw milk sold by one dealer.
2. That circumstantial evidence suggests that this dealer's milk was infected by the quota which he received from A. and possibly C. (*vide* para. 5 below).

* LEVY, E. and JACOBSTHAL, E. *Archiv. f. Hygiene* 1902, Bd. 44, p. 113.

3. That A.'s wife was not accountable for the original infection of milk produced by A.; Mrs. A. and the milk distributed by the dealer were probably infected from the same source.

4. That the source of infection of Mrs. A. and of the milk produced by A. has been determined, but the mechanism of infection is still in doubt. It is not possible definitely to exclude the water of the common well as the vehicle of infection but a review of all the circumstances renders this conclusion very unlikely. The suggestion is made that the connecting link is cows which drank specifically infected water and conveyed the infection in their excreta or milk, but proof of this has not been obtained.

5. If the cow was an intermediary between the infected stream and the milk produced by A., then there is no reason why C.'s contribution to the same dealer's supply should not also have been infected. His cows ran an almost similar risk to A.'s.

6. It is not improbable that I.B., who contracted enteric fever in the year 1934, also owed her infection to the carrier responsible for the present outbreak.

It only remains to add that as soon as it was discovered measures were taken to prevent the specific contamination of the stream.

In conclusion I wish to express my appreciation of the help given in this investigation by the Medical Officers of Health in the affected area.

W. V. SHAW.

List of Appendices.

1. On the method of pasteurization.
2. The lithium experiment.
3. Notes on the clinical aspect, age and sex incidence.
4. Results of examination of the sewage effluent at the point of discharge into the stream.
5. Results of the bacteriological examinations of blood and excreta from the occupants in October, 1936, of the house which was drained to the stream.
6. Results of the bacteriological examinations of the water of the common well at X.
7. Summary of action taken by the Town Councils concerned.

APPENDIX 1.

*Pasteurization.**

All milk distributed by the dealer was first conveyed from the farms to a depot where it was emptied into a common tank; the churns were drained, washed and sterilised by live steam before being used to convey the pasteurized milk to the distributing depot.

The milk is forced under pressure through filters and a steam-heated vessel where it can be raised to the required temperature. The plant has a capacity of some 3,000 gallons per hour. I arranged with the manager that the temperature of the milk under treatment should be raised to 160° F. The result of bacteriological examination of the milk treated by this process was equal to that of a holder process pasteurization at 148° F. for 30 minutes.

I visited in the early hours of the morning and found that the procedure outlined above was followed.

APPENDIX 2.

The Lithium Experiment.

On the 1st November, 1936, the stream was treated at five points with a total quantity of 4 lbs. of lithium citrate. Sample A was taken from the well before the addition of lithium citrate; Samples B and C on one and two days respectively after.

The samples were received on the following dates:—

Sample A received 31.10.36.

Sample B received 2.11.36.

Sample C received 3.11.36.

One litre of each was concentrated and the residue taken up in hydrochloric acid. The final volumes were approximately 2 ml. The hydrochloric acid solution and the residue were examined spectroscopically, with the following results:—

Sample A. Showed the calcium bands very plainly.

Sample B. Showed the calcium bands visible and also an additional band in the red portion of the spectrum. This was identified as a lithium band.

Sample C. Showed a calcium band and also a faint red band of lithium, which was far less intense than the band in Sample B.

* See footnote on p. [6].

APPENDIX 3.

Notes on the Clinical Aspect, etc.

The clinical features varied from a mild attack with little constitutional disturbance to severe prostration with intense intoxication and death. Some of the younger patients had a sharp onset with headache, nausea, pyrexia up to 104° F. to 105° F., and abdominal discomfort: while others showed such evidence of cerebral irritation as stupor, squint and the characteristic cry of meningitis. Some of these cases had a fatal issue, but those who recovered did so with great rapidity and completeness. In several instances there was diarrhoea for the first few days. Early broncho-pneumonia was a feature in some cases and in others the infection was centred upon the genito-urinary system. This complication prolonged the illness for several weeks beyond the initial period of pyrexia.

Four patients had perforation of the bowel and were operated upon; two recovered. Intestinal haemorrhage was also met with in several instances and necessitated in one case transfusion of blood from a suitable donor.

There were a few cases of phlebitis with venous thrombosis during convalescence and one convalescent child died suddenly from pulmonary embolism.

Of the 284 Bournemouth resident patients, 31 died, giving a case fatality of 10·9 per cent., while the corresponding figures for Poole are 205, 17 and 8·3 respectively. There were at Christchurch 29 patients and three fatal cases, giving a fatality rate of 10·3 per cent.

Age and Sex Incidence.

BOURNEMOUTH.

<i>Age.</i>				<i>Male.</i>	<i>Female.</i>
0-5	19	15
6-10	26	28
11-15	21	24
16-20	7	24
21-25	10	15
26-30	13	11
31-35	4	9
36-40	2	9
41+	13	34
				<hr/> 115 <hr/>	<hr/> 169 <hr/>

POOLE.

<i>Age.</i>				<i>Male.</i>	<i>Female.</i>
0-5	15	14
6-10	15	15
11-15	14	15
16-20	15	17
21-30	9	21
31-40	6	20
41+	7	22
				<hr/> 81 <hr/>	<hr/> 124 <hr/>

CHRISTCHURCH.

Age.				Male.	Female.
0-5	2	4
6-10	1	3
11-15	1	2
16-20	1	2
21-25	—	3
26-30	2	1
31-35	1	—
36-40	—	—
41+	—	3
Not ascertained	1	2
				<hr/> 9	<hr/> 20

APPENDIX 4.

Results of the examination of the Sewage Effluent at the point of discharge into the Stream.

Date.	Sample.	Result.	Remarks.
18. 9.36	2	No <i>B. typhosus</i> found.	
21. 9.36	3	No <i>B. typhosus</i> found.	
24. 9.36	4	No <i>B. typhosus</i> found.	
2.10.36	5	No <i>B. typhosus</i> found.	Examined by Professor Wilson
*16.10.36	6	<i>B. typhosus</i> present in large numbers.	„ „
24.10.36	7	<i>B. typhosus</i> present in large numbers.	

Samples taken from above the point of entry of Effluent.

29.9.36	Sample of Stream water	... No <i>B. typhosus</i> found.
29.9.36	Sample from a cesspit	... No <i>B. typhosus</i> found.

Reporting on sample 6 Professor Wilson (Queen's University, Belfast), who has devised an improved method for the isolation of *B. typhosus* in water and sewage, said that there were between 100 and 200 typhoid bacilli in each *ml.* and that the organism was agglutinated up to 1:12,000 or to the full titre of the serum.

Reporting on 27th October on sample 7 Dr. Scott said, " I am practically certain that the sample of sewage effluent received yesterday contains very numerous *B. typhosus*. I have subcultured a few colonies for confirmatory tests to-morrow. The plates are quite different in appearance from those over which I toiled a month or so ago. I don't think I missed typhosus colonies: they simply were not there."

Two days later Dr. Scott wrote, " I am now prepared to say that the effluent sent on 24th October (received here on 26th) contained very numerous *B. typhosus* (approximately 200 per 1 *ml.*) typical in every respect and xylose positive like those isolated in the epidemic in Poole and Bournemouth."

The significance of the last sentence of Dr. Scott's statement on 27th October will be appreciated.

* Suspected inmate absent from 10th August to 28th September.

APPENDIX 5.

Results of the Bacteriological Examination of Specimens of Blood and Excreta from the Occupants in October, 1936, of the house which was drained to the Stream.

Identity number.	Result.			Remarks.
	Faeces.	Urine.	Blood.	
I	Pos.	Neg.	Not examined.	{ Received T.A.B. injection, Sept., 1936. " " " " " " " "
	Pos.	Neg.		
II	Neg.	Neg.		
III	Neg.	Neg.		
IV	Neg.	Neg.		
	Neg.	Neg.		
V	—	—	Neg.	Excreta unobtainable.
VI	Neg.	Neg.	Neg.	
VII	Neg.	Neg.	Neg.	Excreta unobtainable.
VIII	—	—	Neg.	
IX	Neg.	Neg.	Neg.	
X	Neg.	Neg.	Neg.	
XI	Neg.	Neg.	Not examined.	{ Received T.A.B. injection, Sept., 1936. " " " " " " " " " "
	Neg.	Neg.		
XII	Neg.	Neg.		
XIII	Neg.	Neg.		
XIV	Neg.	Neg.		
XV	Neg.	Neg.		
XVI	Neg.	Neg.		

APPENDIX 6.

Results of the Bacteriological Examinations at the Ministry's Laboratory of the Water of the Common Well at X.

2.9.36. Sample 1. (Taken from A.'s tap.)

Count at 37°C.

after 48 hours = 4 per 1 ml.

B. coli.

50 ml.	10 ml.	1 ml.
+	+	—
—	—	—
—	—	—
—	—	—
—	—	—

Probable number of *B. coli aerogenes* in 100 ml. of the original water = 3.

22.9.36. Sample 2. (Taken from A.'s tap.)

Count at 37°C.

after 48 hours = 9 per 1 ml.

B. coli.

50 ml.	10 ml.	1 ml.
+	+	—
—	—	—
—	—	—
—	—	—
—	—	—

Probable number of *B. coli aerogenes* in 100 ml. of the original water = 3.

Two samples direct from the well.

Sample received on Nov. 3rd.

Colony count on agar at 37°C.
29 per 1 ml.

Coli-reaction.

Absent with each of 5 quantities
of 10 ml.

Sample received on Nov. 5th.
(After flooding).

Coli-reaction.

Colony count on agar at 37°C.
20 per 1 ml.

Present in 1 out of 5 quantities of
10 ml.

Probable number of *B. coli* per 100 ml. = 2
(strain isolated = *Bact. coli* faecal type 1).

Attempts to isolate *B. typhosus* failed with both these waters. On bacteriological examination they would both be passed as free from dangerous contamination.

APPENDIX 7.

Action taken by the Town Councils.

Foremost among the measures adopted to prevent spread of the disease was the removal of the patients to hospital, and the major problem which had to be faced was lack of accommodation. At the beginning of the outbreak it consisted of 70 patient-beds at the Bournemouth and 28 at the Poole Isolation Hospital. On 25th August, when the number of persons at risk had been approximately estimated, I advised the Chairman of the Health Committee of the Bournemouth Town Council that it would be necessary to make provision for at least 250 patients. With the co-operation of the Public Assistance Committee and the Board of Management of the Victoria Hospital, Bournemouth, additional accommodation was secured at Fairmile Infirmary and at the Poole Road branch of the Victoria Hospital. Even so it was found necessary to take over a large house, Haddon Hill, and convert it into a temporary hospital with the addition of two temporary ward blocks. The bed accommodation at the Poole Isolation Hospital was increased to 68; 49 beds were provided at the Fairmile Infirmary; 42 at the Poole Road branch of the Victoria Hospital; and 80 at Haddon Hill.

The Medical Officer of Health of Poole promptly realised that an emergency of some magnitude had arisen and at a special meeting of the Health Committee on the 25th August he was given full power to do whatever he thought necessary and in this he had the hearty co-operation of his colleague, the Borough Engineer. Within a few days an old disused ward block at the Hospital was reconditioned and equipped, and by the end of a week a complete new block with proper sanitary accommodation had been erected, equipped, and put into service. This was speedily followed by two other similar blocks, and finally the total number of patient beds provided was 208 with a nursing staff of 68 and a domestic staff of 28. *Pari passu* with the erection of new ward blocks, additions had to be made to the administrative block for staff and enlargement of the kitchen and scullery was also necessary. An operating theatre was provided, electric light was installed, and the requisite enlargement of the sewage disposal plant was carried out forthwith.

At the commencement of the outbreak the Borough of Christchurch was dependent on accommodation provided by neighbouring authorities. Two patients from Christchurch were admitted to the Poole Isolation Hospital, and the remaining 27 were sent to the Salisbury Isolation Hospital.